The compounds of the present invention can be prepared or synthesized according to any suitable methods. Preferably, synthetic methods illustrated in the following Section F are used to prepare the compounds.

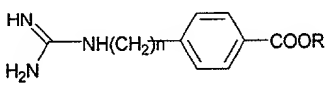
Also preferably, the compound, or its pharmaceutically acceptable salt thereof, is provided in the form of a pharmaceutical composition, either alone or in combination with a pharmaceutically acceptable carrier or excipient.

The compounds of the present invention can be prepared as their pharmaceutically acceptable salts with any suitable acids. For example, inorganic acids, such as hydrochloric acid, hydrobromic acid, nitric acid, sulfuric acid, and phosphoric acid, etc., can be used. In another example, organic acids, such as formic acid, acetic acid, propanoic acid, benzoic acid, maleic acid, fumaric acid, succinic acid, tartaric acid, citric acid, etc., can be used. In still another example, alkyl sulfonic acid, such as methyl sulfonic acid, ethyl sulfonic acid, etc., can be used. In yet another example, aryl sulfonic acid, such as benzene sulfonic acid, p-toluene sulfonic acid, etc, can be used.

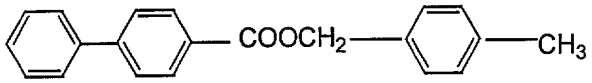
C. Treatment and prevention methods

In another aspect, the present invention is directed to a method for treating or preventing a disease or disorder caused by or associated with bacterial infection, which method comprises administering, to a subject to which such treatment or prevention is needed or desirable, an effective amount of an agent that selectively inhibits bacterial DNA replication initiation, or a pharmaceutically acceptable salt thereof, thereby said disease or disorder is treated or prevented.

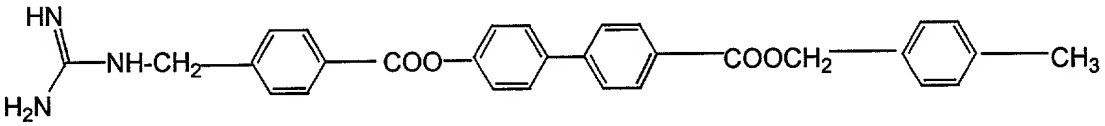
Preferably, the disease or disorder is caused by or associated with *E. coli* or *H. pylori* infection. Also preferably, the disease or disorder caused by or associated with the bacterial, especially, *E. coli* or *H. pylori* infection, is treated or prevented by administering to the subject an effective amount of a compound, or a pharmaceutically acceptable salt thereof, having the following formula II:



wherein n is an integer from 0-1, and R is elected from the group consisting of hydrogen, C_{1-10} alkyl, C_{1-10} aryl and



Any suitable compound or its pharmaceutically acceptable salt thereof, including the ones described in the above Section B, can be used. Preferably, the compound to be administered has the following formula III (NE-2001):



Any subject can be treated by the present method. Preferably, a mammal, and more preferably, a human, is treated by the present method.

The present method can be used to treat or prevent any disease or disorder caused by *E. coli* or *H. pylori* infection. Preferably, the disease or disorder caused by *H. pylori* infection to be treated or prevented is chronic gastritis, gastroduodenal ulcer, adenocarcinoma of the distal stomach, gastric lymphoma or gastric cancer.

The present method can be used to treat or prevent disease or disorder caused by infection of any *E. coli* or *H. pylori* strains. For example, the disease or disorder caused by the following *H. pylori* strains can be treated or prevented:

1: Wang et al., Negative selection of T cells by Helicobacter pylori as a model for bacterial strain selection by immune evasion. J Immunol. 2001 Jul 15;167(2):926-34.

2: Peek RM Jr., . Helicobacter pylori strain-specific activation of signal transduction cascades related to gastric inflammation. J Physiol Gastrointest Liver Physiol. 2001 Apr; 280(4):G525-30.

3: Israel et al., Helicobacter pylori strain-specific differences in genetic content, identified y microarray, influence host inflammatory responses. Clin Invest. 2001 Mar; 107(5):611-20.

4: Vitkute et al., Specificities of eleven different DNA methyltransferases of Helicobacter pylori train 26695. Bacteriol. 2001 Jan; 183(2):443-50.

5: DeLoney and Schiller, Characterization of an In vitro-selected amoxicillin-resistant strain of Helicobacter pylori. Antimicrob Agents Chemother. 2000 Dec; 44(12):3368-73.

6: Hua et al., Isolation of a single strain of Helicobacter pylori from the antrum and body of individual patients. Eur J Gastroenterol Hepatol. 2000 Oct; 12(10):1129-34.

7: Occhialini et al., Distribution of open reading frames of plasticity region of strain J99 in Helicobacter pylori strains isolated from gastric carcinoma and gastritis patients in Costa Rica. Infect Immun. 2000 Nov; 68(11):6240-9.

8: Fassbinder et al., Structural and functional analysis of the riboflavin synthesis genes encoding GTP cyclohydrolase II (ribA), DHBP synthase (ribBA), riboflavin synthase (ribC), and riboflavin deaminase/reductase (ribD) from Helicobacter pylori strain P1. FEMS Microbiol Lett. 2000 Oct 15; 191(2):191-7.

9: Enroth et al., Helicobacter pylori strain types and risk of gastric cancer: a case-control study. Cancer Epidemiol Biomarkers Prev. 2000 Sep; 9(9): 981-5.

10: Petersen et al., Role of strain type, AGS cells and fetal calf serum in Helicobacter pylori adhesion and invasion assays. FEMS Immunol Med Microbiol. 2000 Sep; 29(1):59-67.

11: Matsui et al., Recurrence of gastric ulcer dependent upon strain differences of Helicobacter pylori in urease B gene. Dig Dis Sci. 2000 Jan; 45(1):49-54.

12: Queiroz et al., Factors associated with Helicobacter pylori infection by a cagA-positive strain in children. J Infect Dis. 2000 Feb; 181(2):626-30.

13: Monteiro et al., Lipopolysaccharide structures of Helicobacter pylori genomic strains 26695 and J99, mouse model H. pylori Sydney strain, H. pylori P466 carrying sialyl Lewis X, and H. pylori UA915 expressing Lewis B classification of H. pylori lipopolysaccharides into glycotype families. Eur J Biochem. 2000 Jan; 267(2):305-20.

14: Peek et al., Helicobacter pylori strain-specific genotypes and modulation of the gastric epithelial cell cycle. Cancer Res. 1999 Dec 15; 59(24):6124-31.

15: Aspinall et al., A structural comparison of lipopolysaccharides from two strains of Helicobacter pylori, of which one strain (442) does and the other strain (471) does not stimulate pepsinogen secretion. Glycobiology. 1999 Nov; 9(11):1235-45.